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The Black Death, 1346–1353: The complete history

By Ole J. Benedictow.

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“Historians have generally paid little attention to epidemics other than the Black Death and the Great Plague of London.” So writes John Duffy (1977), referring to events in the fourteenth century and 1665, respectively. Thankfully this has changed somewhat since that was written, but the general historical disinterest in other epidemics has, over the years, funneled scholarship into plague in general and the Black Death in particular. There is an ample supply of books, monographs, articles, and (of course) debates on the Black Death and its role in both social and demographic history. Among the latest arrivals is Ole Benedictow’s *The Black Death, 1346–1353: The complete history*, which combines a valuable tour d’horizon of previous research with some novel mortality estimates.

The Black Death

The Black Death was an epidemic that killed upward of one-third of the population of Europe between 1346 and 1353 (more on proportional mortality below). The precise specification of the time span, particularly the end dates, varies by a year or so, depending on the source. A less severe (but still potent) follow-on epidemic in 1361, ostensibly of the same disease, is, by convention, separate from the Black Death. A common misconception is that black refers to skin discolorations accompanying the disease. Black is meant in the metaphorical sense of terrible. In fact, the term “Black Death” was not used until the middle of the sixteenth century. Contemporaries called it the “pestilence”.

The historical importance of an event that killed such a huge proportion of Europe requires little elaboration. Even by contemporary standards, the Black Death was shocking. Certainly, life in the fourteenth century was short from a modern perspective, but even

the worst mortality events in the thirteenth and fourteenth centuries, up to 1346, do not compare to the Black Death. However, it is important to bear in mind exactly what these mortality crises were during the end of the high middle ages, and in the early period of the late middle ages up to the Black Death. The 1290s witnessed numerous wheat failures throughout Europe, caused in the main by unfavorable weather, and the agricultural situation did not improve in the early fourteenth century. Famine mortalities reached ten percent in some localities. There are even reports by chroniclers of cannibalism, though these are regarded as apocryphal by some historians.

Historians debate whether these stresses represented a true long-run Malthusian crisis. The counter-argument is that medieval agriculture was capable of feeding Europe, meteorological bad luck aside. In any case, the hypothesis that the Black Death itself was an inevitable consequence of population pressure — that the Black Death was endogenous, if you will — is no longer well-regarded. The intercession of some external pathogen is now regarded as a condition without which the Black Death would not have occurred. Just what that pathogen was, and from where it came, are debated to this day (cf. below).

Apart from the second plague (1361), the closest thing to a repeat of the Black Death was the Great Plague of 1665, which by some estimates killed fifteen to twenty percent of the population in certain locales. In modern times, the 1918–19 influenza pandemic comes to mind, and it killed more people than the Black Death because it was truly worldwide and because the twentieth century had much larger population denominators than the fourteenth century. The 1918–19 flu killed perhaps 2.5 percent of the world population — for percentage mortality it doesn't even come close to the Black Death. These comparisons are somewhat arbitrary, as the Black Death struck Europe and western Asia, while the flu was global, but it's safe to say that the world has not experienced anything quite like the Black Death since the fourteenth century.

Although the historical importance of the Black Death is clear, its historical significance — viz., the impact — remains a matter of fierce debate. The story is that the Black Death's demographic wake relieved population pressure and caused the transition out of the

late middle ages. David Herlihy's posthumous volume *The Black Death and the transformation of the west* (1997) advocates this view eloquently and succinctly. The Black Death "broke the Malthusian deadlock that [eleventh and twelfth century] medieval growth had created and which might have impeded further growth in different forms" (Herlihy, p. 38). With the labor equation turned in their favor, peasant serfs would evolve into the yeoman farmers of the fifteenth century.

Others hold that the medieval European social-demographic system rebounded from the Black Death. It was not the wake of an epidemic, according to this view, but the cultural stirrings of the Renaissance and other factors (themselves not a direct result of the Black Death), that lead to the transition from the late medieval period to early modern times. Cantor (2001) deems the causal relationship between the Italian Renaissance and the Black Death "tenuous". Economic demographers David Bloom and Ajay Mahal (1997) provide a quantitative view against the Black Death as a pivot. They estimate that post-pestilence changes in real (wheat-price deflated) wages in England and France do not deviate significantly from the long-term trend. According to Bloom and Mahal, various changes (population, wages, prices) acted in compensatory ways, as best as can be discerned from the scant available evidence. The debate on the impact of the pestilence will continue because the relevant counterfactual — no Black Death — introduces an infinity of alternate universes, with diverse factors such as Malthusian pressure, crop failures, shifting fortunes in the Hundred Years War, and so on, all coming into play.

Was it plague?

The historiography of the Black Death is chock full of debates, none more heated of late than the question of etiology. Plague has been used as general term for any great epidemic, but it is also a specific disease caused by the bacterium *Yersinia pestis*, named after the French bacteriologist Alexandre Yersin (previously called *Pasteurella pestis*, after Yersin's employer, the Institut Pasteur). Like many diseases, plague is a zoonosis: it comes to humans from animals. Plague has a natural reservoir among wild rodents, and a vector in fleas. Plague

persists to this day. Plague foci, as enzoötic regions are called, exist throughout the world, including in Asia, Africa, South America, and the southwestern United States.

Plague ecology is complex, but a thumbnail sketch is that when humans become inserted into the rodent-flea-rodent cycle of *Yersinia pestis* transmission, an outbreak occurs. Although plague exists in a wide variety of rodents including squirrels and marmots, rats are indicted in the Black Death because of their tendency to nest around humans and to stowaway on ships. The black rat (*Rattus rattus*) in particular was the culprit. The brown rat (*Rattus norvegicus*) now prevalent throughout much of the world was, by most accounts, not present in Europe in medieval times. The rat flea, *Xenopsylla cheopis*, is the disease vector. When the rats die of plague, hungry fleas go looking for blood meals elsewhere, and they find humans. On longer voyages, provided the ambient humidity is not too low, the rat flea can survive for a time after the rats have died, persisting on grain dust. There is (again) a debate about whether the human flea, *Pulex irritans*, also transmitted the plague from person to person during the Black Death.

The epidemiology of plague is inexorably linked to rodent and human ecology. Normally, plague bacteria live in small concentrations in the blood of rodent hosts and in the guts of fleas who feed upon them. It is not crystal clear what triggers an epizoötic, but it happens when a chain of biological events are set in train. First, a higher concentration of bacteria accumulate in a rodent. This causes a flea to ingest more bacteria, which multiply in the gut of the flea so much that they subsequently form a solid mass in the flea's stomach. The flea in this state is said to be "blocked". The blockage causes a flea to regurgitate while it feeds, and the regurgitation includes bit of the blockage and therefore has an ultrahigh concentration of bacteria. At these high concentrations, rodents succumb to the plague. When the rodent dies the flea moves on to a new rodent. When an entire rat colony has died, fleas will move onto other animals, including farm animals (except horses, whose smell repels even fleas) and humans. Plague epidemics are thus preceded by epizoötics.

The most common form of plague disease in humans is bubonic plague, characterized by buboes, or enlargements of the lymph nodes, usually in the in the groin, armpits, or

neck, depending upon the location of the flea bite. There is one bubo in most cases, arising 3-5 days after the bite. Today plague is treatable with antibiotics, but untreated, plague case fatality in modern populations is 50–60%. In fatal cases, plague bacteria escape the bubo and enter the bloodstream, killing the victim on average eight days after the bite. A second type of clinical classification is pneumonic plague, which occurs when the bacteria spread to the lungs and cause a pneumonia. Primary pneumonic plague refers to infections acquired from other cases of pneumonic plague (i.e. person to person spread without fleas). Yet another debate about the Black Death surrounds the relative importance of pneumonic versus bubonic plague. A third type of plague, septicemic, occurs when the flea hits a vein and injects plague bacteria right into the bloodstream — septicemic plague essentially skips the step with the buboes, and without prompt treatment it is almost always fatal.

Linking historical disease outbreaks to established modern syndromes is a preoccupation of historical epidemiologists. A repetitive parlor game involves assigning a disease to the Plague of Athens (430 BC), to which Thucydides famously attributed a panoply of diverse symptoms. Medieval accounts of symptoms were more often than not vague, and linkage with modern diseases is full of potential pitfalls. To wit, Creighton (1891) is dubious about the prevalence estimates of leprosy in medieval Britain — it is not clear if most inmates in Europe's numerous leprosaria actually had leprosy as defined by modern medicine (i.e. *Mycobacterium leprae* infection). Another example is that the diagnostic distinction between smallpox and measles — two major diseases — was not made by western medicine until the sixteenth century.

The classic account linking yersinial plague and the Black Death goes like this. The symptoms described by chroniclers agree, *grosso modo*, with the symptoms of modern yersinial plague, particularly the presence and location of boils, interpreted as buboes. The chroniclers were not doctors, however, and as noted most accounts were vague. Boccaccio's novel *Decameron* also offers a description of symptoms, but such is the state of Black Death nosography that we are dependent upon a work of fiction as much as anything else. The apparent spread of the Black Death along shipping routes is congruent with plague, as the black rat

is a good climber and would have accessed ships in harbor via mooring ropes. But many diseases tend to spread along trade routes. Gaps in the story include absence of comments about dead rodents in the chronicles, even though there must have plenty of these if the Black Death were yersinial plague. Possibly, dead rats were not deemed worthy of record. The Black Death also appears sometimes to have traveled faster than one would expect from modern plague epidemiology.

A recent argument in favor of the classic, yersinial, account surrounds detection of plague DNA in dental pulp from teeth in a medieval mass grave in France, ostensibly of Black Death victims (Raoult et al. 2000). The modern DNA test (PCR, polymerase chain reaction) is highly sensitive, which has raised objections about the possibility of false positives. While no single piece of evidence is utterly conclusive, it seems to me the DNA evidence deserves a lot of weight. Even though the grave is not dated precisely to the Black Death, it still puts *Yersinia pestis* relatively close, time-wise, to the scene of the crime. Also, without placing blind faith in lab tests, it still seems to me that the measurement error of PCR is likely to be far smaller than what might be called “speculative error” when there is a free-for-all among hypotheses competing to knock *Yersinia pestis* out of the record books.

The opening salvo in the revisionist, non-yersinial, account was Twigg’s 1984 monograph, *The Black Death: A biological reappraisal*. Twigg suggests anthrax, caused by the spore-forming bacterium *Bacillus anthracis*, as the etiologic agent, while the ensuing debate has also suggested hemorrhagic viral fever — a fourteenth century Ebola — as a possibility. Shrewsbury (1970) argues that a co-epidemic of yersinial plague and louse-borne typhus was responsible for the great mortality of the fourteenth century. The debate is spirited. Scott and Duncan (2001) list a twenty-item bill of particulars against *Yersinia pestis* (pp. 356–362). One theme is that the dynamics of the Black Death are not plague-like, based on reasoning from mathematical models.

Twigg (1984) makes use of John Brownlee’s modeling of plague outbreaks over four centuries. Brownlee was a key figure in the development of epidemiology, but I take his models with a grain of salt. As Fine (1979) discusses in great detail, Brownlee’s notions

of the shape of epidemic curves were underpinned by the incorrect view that all epidemics fall due to diminished virulence. Scott and Duncan (2001) note that the Black Death did not behave according to Reed-Frost dynamics, as a plague epidemic ought to, according to them. On the other hand, Christakos et al. (2005), who likewise provide a strident argument against yersinial plague, fit a series of modified Reed-Frost models to the Black Death. The key lies, evidently, in the modification of the models. These are treacherous waters to enter but despite my own interest in disease modeling more generally, I find the Black Death modeling exercises to be the least persuasive. Models need data, for parameter estimation and for validation. The Black Death data are of especially dubious quality, which permits a wide latitude of possibilities and interpretations when it comes to model fitting.

Different diseases spread differently, and knowing the cause of the Black Death affects many things; it is not unimportant. For one thing, estimates of the speed of spread of the epidemic depend in part on the incubation time of the disease in animal reservoirs or in humans or both. The arrival of the Black Death in a given locale dates from the onset of symptoms minus the incubation period(s). Without an estimate of the duration of incubation, the dynamics become considerably fuzzier. Nonetheless, a point worth emphasizing is that one may study the Black Death and its social and demographic upheavals without committing to linking the causative pathogen or pathogens with any specific germ or germs as we know them today. Clearly, some infectious disease roiled Europe in the mid-fourteenth century, and it would be a pity if scholarship on the Black Death should become too distracted by debates over the cause. Many historians of the Black Death have, quite reasonably, adopted an agnostic stance vis-à-vis the etiologic agent. Readers interested in the debate should also confer Theilmann and Cate (2007).

The complete history

This brings us to *The Black Death, 1346–1353: The complete history*, one of the most demographically-oriented of all the recent histories of the pestilence. Ole Benedictow is an historical demographer and professor of history, emeritus, at the University of Oslo. The subtitle of

the book, *The complete history*, is sure to make Black Death specialists everywhere bridle, but he assures the reader that the book is not intended to be the last word on the subject, but rather “a general synthetic study of the Black Death’s epidemiology, territorial spread and mortality” (p. xi).

The book is divided into five parts. Part one, “What was the Black Death?”, gives a series of reviews of plague and the Black Death. Part two, “Spread of the Black Death”, is a valuable tour of the existing literature, with each country or region in its own chapter. This is the strongest part of the book. It distills literature from diverse sources on the geographic spread of the Black Death. Part three, “Patterns and dynamics of the Black Death”, contains a single chapter that builds upon part two by considering several conundrums of Black Death epidemiology. Benedictow attributes the sometimes seemingly impossibly rapid spread of the Black Death to “metastatic leaps” of fleas moving on ships. Part four, “Mortality in the Black Death”, gives another region by region assessment, this time of death rather than spread. Part five, “The Black Death: Its impact on history”, is a single chapter giving some broader context. Benedictow shares the assessment that the Black Death was a pivot, and it is somewhat curious that he does not cite Herlihy (1997). Social-historical topics, as the Black Death’s role in provoking persecution of Jews are discussed also.

As noted, *The complete history* has a demographic orientation, with the caveat that medieval population studies are a data-starved endeavor, and quantitative studies of this period necessarily involve a lot of assumptions. To his credit, Benedictow discusses the assumptions he uses, both throughout the book and in methodological sections. Readers of the book with a strictly historical interest will thus likely find chapter 26, “The medieval demographic system”, somewhat esoteric, with its discussion of the level 4 Model West life table (life expectancy 25 for women and slightly higher for men) and so on. Those with no background in demography will likely not find enough information to appreciate fully the intricacies of the life table. But it’s hard to fault Benedictow for keeping the formal demography exposition to a minimum; it’s a book on the Black Death, after all, not a demography

textbook. In any case, the audience of *Population and Development Review* will feel right at home in this chapter.

The book has some small inconsistencies. For example, on p. 5, “the Black Death was the *first* and particularly violent outbreak [of plague]” (emphasis added) — Benedictow, evidently, does not believe that the so-called Plague of Justinian (541 AD) was caused by plague. Clarification comes thirty-four pages later: “The Black Death was not the first clearly identified wave of plague epidemics...”, and he goes on to discuss the Justinianic outbreak. The former instance apparently refers to the Black Death as the epidemic that ushered in the second plague pandemic, while the latter deals with all of history. The misunderstanding could be avoided if the various overviews that open the book were better organized.

I did not especially care for the repeated military metaphor, with plague having “armies” and “strategy”, and making “campaigns”, “invasions”, “bridgeheads”, and so forth, though this is a question of taste. For the most part, Benedictow writes clearly and gets his meaning across. The book’s region by region organization gives it the feel, somewhat, of a stamp collection. Regional specialists will doubtless have their cavils, but the breadth provided by stamp collections make them extremely useful as encyclopedia-like tomes. When I want to know what has been done on the Black Death in a given region of Europe, I certainly will pull Benedictow’s book off the shelf.

The more serious problem with the book has to do with the way it presents various perspectives. Benedictow firmly believes that the Black Death was yersinial plague. Some will disagree; so be it. But I think Benedictow has done his readers a disservice by not giving the other side a fairer hearing. Right or wrong, Twigg’s 1984 monograph is a serious work, not a conspiracy theory. Certainly, Black Death experts are currently divided into camps. Twigg deserves a footnote, at least.

Benedictow places the geographic origin of the Black Death in the Golden Horde, dismissing theories of a far eastern origin. This is not a settled matter, either. William H. McNeill’s influential (and controversial) book *Plagues and peoples* (1976) places the origins of the Black Death much farther east; he does not pinpoint an epicenter but offers several

eastern scenarios. Most studies of the Black Death in the yersinial mold are in concordance that it came from the east. The outbreak at Caffa (or Kaffa, modern-day Feodosiya) in the Crimea indicates that the Black Death was in the Golden Horde by 1346.

The Caffa outbreak has become the stuff of legend. The Genoese established a trading colony at Caffa. A street brawl escalated into a small war, with a Tatar army eventually laying siege to the city. Then the Black Death erupted among the Tatars, devastating the besiegers. Desperate, the Tatars catapulted dead bodies into the citadel, spreading the Black Death to the Genoese. The besieged then beat a retreat to Genoa via Constantinople and Messina, thus spreading the Black Death to Asia Minor, Sicily, and continental Europe. Or so the story goes. Clearly, the Black Death passed through the Golden Horde — but did it start there? The uninitiated reader is not given a sufficiently distinct notion that the Golden Horde is not a consensus. Interested readers should also consult a classic debate between Norris (1977, 1978) and Dols (1978) (cf. also Schamiloglu 1993).

The most original contribution of the book is part four, a series of mortality estimates, including, in chapter 33, an extrapolation of these estimates to a novel synthetic estimate for Europe as a whole. Benedictow is suitably alert throughout to “Problems of Source Criticism, Methodology, and Demography” (chapter 27). I am concerned, though, that he seems to be especially source-critical when it comes to data that do not fit into his picture of high mortality. Benedictow’s correctly asserts that wills may be used to date the arrival of the Black Death in a locale, but are less useful in judging mortality, since wills were composed as a fearful reaction to the sudden increase in death, even in cases where those writing the wills would ultimately survive. However, he notes changing intervals between dating and probate (p. 137), which would seem to indicate that, at least in some jurisdictions, probate data are available. This is especially relevant for London, where no contemporary ecclesiastical registers survive (p. 135).

To me, the most striking example of Benedictow eschewing data that did not fit his story was his consideration of Black Death mortality in Mallorca in the Balearic Isles. It provides a compact example of his approach. Benedictow reviews the available evidence,

which points to sixteen percent island-wide mortality (p. 280). He notes that this mortality is low in comparative perspective. Then he comments, “so far no scholar has come up with any epidemiologically relevant reason(s)” for the good fortunes of Mallorca (p. 281). He observes that the balance of the rural and urban mortality does not fit the typical pattern. Then the sixteen percent figure is summarily set aside: “these Mallorcan data are, therefore, infested with major problems of demography, sociology and source criticism”. That there should be regional differences in mortality strikes me as completely normal for any epidemic. We see this in disease outbreaks down to this day. Sixteen percent mortality may indeed be an exception, but many distributions have thick tails and I fail to see why exceptions are unexpected. All the more since Mallorca is an island. The rural-urban difference may be viewed as data problems or as consistent with outlier status, depending on how one looks at it. My fear is that the baby has gone out with the bathwater.

After sorting (and sifting) the available evidence, Benedictow’s synthetic estimate for Black Death mortality in Europe as a whole is 60 percent (p. 383). This is based on “the remarkable similarity of the levels of mortality in . . . widespread and diverse regions” (p. 381). Here, he duly notes that previous estimates are in the 25–33 percent range. If Benedictow is correct, and assuming a European population of circa 80 million, then the Black Death would about equal the 1918-19 flu in absolute die-off. Crisis mortality is intrinsically hard to get at — the exact number dead in the twentieth century event is by no means settled, either (cf. Johnson and Mueller 2002). Benedictow’s estimates for mortality from the Black Death alone are on par with what previous experts have come up with for the Black Death and the 1361 plague combined, or, in some cases, also including the famine mortality earlier in the fourteenth century.

Benedictow’s mortality estimates may eventually come to be regarded as the standard, in spite of doubts that the remarkably similar die-off across regions is due in part to rejecting less severe data through source criticism. The estimates are internally consistent with his assessments of plague case fatality (circa 80 percent, p. 350) and prevalence. If plague lethality is over 50 percent in modern populations, then 80 percent is not implausible for

medieval times, considering the nutritional stress of the fourteenth century. The intervening six hundred years is also enough time for *Yersinia pestis* (or another pathogen) to have evolved to lower virulence. The higher the case fatality, the lower the prevalence need be (*ceteris paribus*) to produce a given total mortality. So Benedictow's estimates do not require that everyone still standing at the end of the day was a plague survivor in the literal sense (which would seem unrealistic). What is more, one of the ironies of the middle ages is that even though most of the population was rural, they lived, at the microlevel, with very little elbow room. Benedictow's estimates are consistent with this, since if one person in an abode got plague, the likelihood of everyone getting it was great. I retain my doubts that the Black Death by itself killed greater than 50 percent of Europe, but I do not dismiss it entirely.

Benedictow describes plague as a "hydra-headed monster", and I hope I have not underestimated the Herculean task Benedictow faced in taming this beast for the printed page. This is a demographic history of the Black Death, not a general or social history. Historical context is important, but plague is also a biological and demographic topic, and we need books that focus strictly on the historical epidemiology. For one thing, it's hard for a single author to balance everything. The late Norman F. Cantor was a medieval historian at New York University, and clearly he was not ignorant of the socio-historical milieu of the late middle ages. But Cantor's *In the wake of the plague: The black death and the world it made* (2001) unfortunately is poorly-researched when it comes to purely epidemiological aspects. One example is that Cantor gives serious consideration to astronomer Fred Hoyle's loony notion that the Black Death came from outer space. Benedictow has stuck mostly to historical demography, giving a round-up of the available quantitative evidence for all of Europe, and by putting all of this in one place he has performed a valuable service.

Scholars looking for an entrée to the Black Death could consult Benedictow's book as a country by country field guide to the demographic history. For a general introduction to the Black Death, I think Gottfried (1983) remains a better overview, despite its publication date. It gives more emphasis to the Black Death's place in medieval history; for better or for worse it has less demography. As with Benedictow's book, some will dispute the yersinial

outlook in Gottfried, but so far at least, I think the book has weathered well. Gottfried's is an environmental history, and the influence of McNeill and Alfred Crosby is palpable. A more standard treatment is Ziegler (1969), older but still in-print. Some will quarrel that these books are out-of-date but for those wishing to get up-to-speed on the Black Death from a knowledge base of zero, they are not a bad place to start.

Every library that covers population studies or epidemiology or medieval history should have a copy of Benedictow's book, and any historical demographer whose interest extends prior to early modern history will want a copy as well. The book is too specialized to be used as a textbook, apart from in advanced graduate seminars on the Black Death, but to be fair it does not seem to have been written with the classroom in mind.

Despite all the existing scholarship on the Black Death, there is still room for more work on the great mortality crises of the fourteenth century. The second plague epidemic, beginning in 1361, is not covered by Benedictow, and although mortality was lower than in the Black Death, it still killed some ten percent of Europe by standard estimates. I have no quarrel with Benedictow sticking to the Black Death proper — the second epidemic merits a volume of its own. There are a number of opportunities for research on the two outbreaks in comparative perspective: the later epidemic appears to have skewed younger, which may have been a cohort effect of acquired immunity. If so, this would have implications on Black Death attack rates and hence on case fatality. Iceland was affected by the second epidemic (though not until the early fifteenth century) but escaped the Black Death, which may provide a natural experiment of sorts, especially since the second epidemic struck Iceland severely (Tomasson 1977).

In sum, Benedictow's *The Black Death, 1346–1353: The complete history*, is a useful compendium of the demography of the most calamitous epidemic in documented history. With its region by region organization, it is an excellent go-to guide of the demography of the Black Death. It would have been improved by giving a fuller account of opposing views, and many of Benedictow's estimates, particularly of mortality, will need to be digested more

fully by other scholars before they may be regarded as authoritative. The book's value will be greatest for experts who are able to weigh the arguments critically.

It is typical to close a review such as this with the sentiment that "I hope this book will spur more work on this subject", but in the case of the Black Death, this is clearly unnecessary, as it is the most crowded area of historical epidemiology. Nonetheless, there is room for more work on the Black Death and I hope that historical demographers and epidemiologists will never stop looking afresh at the available data.

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